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L5	(gp39 or cd40L or cd40 adj ligand or cd154 or 5c8) and (treat$ or therap$ or inhibit$ or suppress$ or prevent$ or administ$)same (atherosclerosis)	278	L5
L4	(gp39 or cd40L or cd40 adj ligand or cd154 or 5c8) same (atherosclerosis)	41	L4
L3	(gp39 or cd4oL or cd40 adj ligand or cd154 or 5c8) and (atherosclerosis)	274	L3
L2	yellin-michael$	12	L2
L1	yellin-michael	0	L1

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Term	Documents
GP39.DWPI,EPAB,JPAB,USPT,PGPB.	215
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CD40LS.DWPI,EPAB,JPAB,USPT,PGPB.	2
CD40.DWPI,EPAB,JPAB,USPT,PGPB.	1780
CD40S	0
LIGAND.DWPI,EPAB,JPAB,USPT,PGPB.	71876
LIGANDS.DWPI,EPAB,JPAB,USPT,PGPB.	49822
CD154.DWPI,EPAB,JPAB,USPT,PGPB.	228
CD154S.DWPI,EPAB,JPAB,USPT,PGPB.	1
5C8.DWPI,EPAB,JPAB,USPT,PGPB.	95
((GP39 OR CD40L OR CD40 ADJ LIGAND OR CD154 OR 5C8) SAME (ATHEROSCLEROSIS)).USPT,PGPB,JPAB,EPAB,DWPI.	41

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L4	(gp39 or cd40L or cd40 adj ligand or cd154 or 5c8) same (atherosclerosis)	41	L4
L3	(gp39 or cd4oL or cd40 adj ligand or cd154 or 5c8) and (atherosclerosis)	274	L3
L2	yellin-michael$	12	L2
L1	yellin-michael	0	L1

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L4: Entry 21 of 41 File: USPT Jan 22, 2002

DOCUMENT-IDENTIFIER: US 6340459 B1
TITLE: Therapeutic applications for the anti-T-BAM (CD40-L) monoclonal antibody 5C8 in the treatment of reperfusion injury in non-transplant recipients

Detailed Description Text (49):
In embodiments of this invention the condition dependent on CD40 ligand-induced activation of endothelial cells is selected from the group consisting of atherosclerosis, reperfusion injury, allograft rejection, organ rejection, and chronic inflammatory autoimmune diseases.

Detailed Description Text (50):
In a specific embodiment the atherosclerosis is accelerated atherosclerosis associated with organ transplantation. In situ CD40 and CD40L expression in accelerated atherosclerosis associated with transplant rejection have been studied. Frozen sections of coronary arteries from 4 heart transplant patients that required retransplantation due to accelerated atherosclerosis were analyzed by routine immunohistochemistry utilizing anti-CD40 mAb G28.5, anti-CD40L mAb 5C8 or control mAbs. Routine H & E staining revealed the typical intimal hyperplasia, smooth muscle cell proliferation, and inflammatory cell infiltration associated with the disease. CD40 was widely expressed in the lesions: endothelial cells, foam cells and infiltrating inflammatory cells all express CD40. CD40L immunoreactivity was observed as discrete, faint staining of infiltrating mononuclear cells, presumably CD4+ T cells. Together, these studies demonstrate the presence of CD40L+ mononuclear cells and CD40+ endothelial cells, foam cells, and inflammatory cells in situ in lesions of accelerated atherosclerosis associated with transplantation.

Detailed Description Text (54):
This invention provides a method of treating a condition dependent on CD40 ligand-induced activation of macrophages in a subject, comprising the above-described method of inhibiting activation of macrophages by CD40 ligand in a subject. In specific embodiments, the condition dependent on CD40 ligand-induced activation of macrophages is atherosclerosis or rheumatoid arthritis.

Detailed Description Text (135):
Finally, endothelial cells are activated in a variety of diseases mediated by CD4.sup.+ T cells. For example, endothelial cell surface adhesion molecules are upregulated in rheumatoid arthritis (62), scleroderma (63) and in transplant rejection (64). In addition, CD4.sup.+ T cells play roles in atherosclerosis (65) and accelerated atherosclerosis associated with transplantation (60). The precise mechanistic role of CD40L mediated interactions with endothelial cells in these diseases is not known. However, an antibody to CD40L, MR1, inhibits murine models of diseases mediated by CD4.sup.+ T cells and/or inflammatory cell infiltrates. For example, MR1 prevents the synovial lining cell hypertrophy and cellular infiltrate associated with collagen-induce arthritis, a murine model of rheumatoid arthritis (66). Moreover, MR1 inhibits a murine model of multiple sclerosis (EAE) and inhibits allograft rejection (67). Blocking CD40L dependent interactions with endothelial cells and/or fibroblasts mediates, in part, these effects of MR1. The results disclosed herein suggest that CD40L-CD40 interactions on the surface of endothelial cells play immunopathogenic roles in inflammatory diseases.

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CD154S.DWPI,EPAB,JPAB,USPT,PGPB.	1
5C8.DWPI,EPAB,JPAB,USPT,PGPB.	95
((GP39 OR CD40L OR CD40 ADJ LIGAND OR CD154 OR 5C8) AND (TREAT$ OR THERAP$ OR INHIBIT$ OR SUPPRESS$ OR PREVENT$ OR ADMINIST$)SAME (ATHEROSCLEROSIS)).USPT,PGPB,JPAB,EPAB,DWPI.	278

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L4	(gp39 or cd40L or cd40 adj ligand or cd154 or 5c8) same (atherosclerosis)	41	L4
L3	(gp39 or cd4oL or cd40 adj ligand or cd154 or 5c8) and (atherosclerosis)	274	L3
L2	yellin-michael$	12	L2
L1	yellin-michael	0	L1

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LIGANDS.DWPI,EPAB,JPAB,USPT,PGPB.	49822
CD154.DWPI,EPAB,JPAB,USPT,PGPB.	228
CD154S.DWPI,EPAB,JPAB,USPT,PGPB.	1
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